

Alcohol Withdrawal & Delirium Tremens

A plain-English guide for you and your family

WHAT IS HAPPENING

Your brain uses two main chemicals to stay balanced: GABA (the brain's main calming signal) and glutamate (the brain's main wake-up signal). When someone drinks heavily for weeks or months, alcohol forces GABA to work overtime and tells glutamate to quiet down. The brain adapts to this new normal by pulling some GABA receivers off the surface of nerve cells and adding more glutamate receivers. But when alcohol suddenly disappears — usually 2 to 4 days after the last drink — the brain's balance snaps in the opposite direction: GABA collapses and glutamate floods the system like a gas pedal stuck to the floor. This electrical storm causes confusion, agitation, seeing or hearing things that are not there, shaking, drenching sweats, racing heart, high blood pressure, and fever. Every symptom is the body trying to survive what the brain senses as a life-threatening crisis. Muscles can break down, spilling their insides into the blood. Kidneys can fail. The heart can beat in dangerous rhythms. Seizures can start and not stop. This is why delirium tremens is a medical emergency: without the right treatment — benzodiazepines (medicines that restore GABA's calming effect), fluids, salts like potassium and magnesium that keep the heart beating right, and thiamine (vitamin B1 that brain cells need to turn sugar into energy) — the window closes fast.

What you might feel

- Confusion and disorientation
- Agitation and combativeness
- Visual, auditory, or tactile hallucinations
- Coarse tremor (shaking)
- Profuse sweating
- Fast heart rate (often over 100–120 beats per minute)
- High blood pressure (top number often over 160 mmHg)
- High fever (100.4–104°F or 38–40°C)
- Generalized tonic-clonic seizures (whole-body convulsions)
- Insomnia and severe sleep disturbance

Call the doctor right away if...

- Temperature above 102.2°F (39°C) or continuing to rise despite cooling measures—signals severe autonomic instability and high risk of organ failure.
- New-onset seizures or any seizure lasting longer than 5 minutes—warns of possible progression to status epilepticus (a seizure that will not stop), a brain emergency requiring immediate intervention.
- Systolic blood pressure over 180 mmHg or heart rate over 140 beats per minute despite benzodiazepines—means sedation is inadequate and the patient is at high risk for stroke, dangerous heart rhythms, or cardiovascular collapse.
- Worsening confusion or decreased responsiveness after starting treatment—may indicate a second problem such as infection, head injury, hepatic encephalopathy (liver failure causing brain toxicity), or Wernicke encephalopathy from thiamine deficiency.
- Dark (tea-colored) urine, rising creatinine in blood tests, or tender, swollen muscles—suggests rhabdomyolysis, where muscle is breaking down and releasing toxins that can shut down the kidneys.

What the care team will do

- Start continuous pulse oximetry, cardiac telemetry (heart rhythm monitoring), and automated blood pressure checks every 15 minutes until the patient is stable.
- Administer IV thiamine 500 mg slowly over 30 minutes before or at the same time as any IV dextrose (sugar) solution.
- Give IV benzodiazepines—lorazepam 2 to 4 mg or diazepam 5 to 10 mg—every 5 to 15 minutes using CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised) scores to guide dosing until tremor stops, heart rate drops below 100, and the patient is calm but arousable.
- Place the patient in a quiet, well-lit room with constant observation; remove any objects that could be used for harm; use calm verbal redirection and avoid physical restraints unless the patient is an immediate danger to self or staff.
- Check core temperature every hour and use cooling measures—remove blankets, apply ice packs to the groin and armpits, give IV fluids—if temperature rises above 101°F (38.3°C).
- Draw blood for glucose, electrolytes (sodium, potassium, magnesium, phosphate), liver function, and complete blood count; correct magnesium below 2.0 mg/dL and potassium below 3.5 mEq/L immediately.
- Keep IV access secure with at least one large-bore (18-gauge or larger) peripheral IV or consider a central line if the patient is combative or veins are poor; flush the line every 4 hours.

- Assess for aspiration risk before giving anything by mouth; keep the patient NPO (nothing by mouth) or give only small sips if alert enough to protect the airway; keep suction equipment at the bedside.

Medicines you may be given — and why

Benzodiazepines (lorazepam or diazepam)

When someone drinks alcohol every day for weeks or years, the brain changes to adapt. The brake system—GABA-A receptors, which calm brain cells—turns down. The gas pedal—glutamate NMDA receptors, which speed up brain cells—turns up. When alcohol suddenly stops, the brain is left with weak brakes and a floored gas pedal. Brain cells fire wildly. This wild firing causes seizures, confusion, seeing things that are not there, a racing heart, high blood pressure, fever, and sweating. Benzodiazepines latch onto GABA-A receptors and make the remaining GABA work much stronger. This restores the brake and slows the dangerous overactivity. Lorazepam (Ativan) or diazepam (Valium) is given through an IV—a tube into a vein. Start with 2 to 4 milligrams every 5 to 15 minutes. Keep going until the heart rate, blood pressure, sweating, and agitation come down. The goal is to calm the autonomic storm—the overactive nervous system controlling heart and blood vessels—and prevent seizures.

Phenobarbital

Phenobarbital is a backup when benzodiazepines alone cannot control severe withdrawal. It works at two sites. First, it directly opens the chloride channel on GABA-A receptors—even without GABA present. Second, it blocks glutamate NMDA receptors, the gas pedal. This double action calms the brain more deeply. Phenobarbital also lasts much longer in the body—half-life 80 to 120 hours—so one dose keeps working for days. It is given as a loading dose, 10 to 20 milligrams per kilogram of body weight. This is divided into smaller IV doses over an hour. Then it is maintained with daily dosing if needed. Because it works even when GABA is scarce, it can rescue patients who are refractory—not responding—to benzodiazepines.

Propofol

Propofol is used in the ICU for the sickest patients—those whose withdrawal is so severe that benzodiazepines and phenobarbital together are not enough. It binds to GABA-A receptors and greatly amplifies chloride flow. The result is deep sedation or even general anesthesia. Propofol is given as a continuous IV drip, starting at 5 to 10 micrograms per kilogram per minute. Then it is adjusted up or down, so it can be changed quickly. It works within seconds and wears off within minutes when stopped. This gives very tight control. The patient usually requires a breathing tube—endotracheal intubation—because propofol can completely suppress spontaneous breathing.

Thiamine (vitamin B1)

Chronic alcohol use drains thiamine because alcohol blocks its absorption in the gut. The liver cannot store much. Thiamine is required for three key enzymes that turn glucose into energy inside the mitochondria—the power plants of cells. Without thiamine, brain cells—especially in the thalamus (the brain's sensory relay station), mammillary bodies (small round structures at the base of the brain involved in memory), and brainstem—run out of energy and die. This causes Wernicke encephalopathy: confusion, eye movement problems, and unsteady walking. If untreated, this leads to Korsakoff syndrome—permanent memory loss. Thiamine 500 milligrams IV three times daily for at least 3 days replaces the shortage before glucose is given. Giving glucose without thiamine forces the brain to try to burn it without the needed helper. This can trigger or worsen Wernicke encephalopathy. After 3 days, switch to 100 milligrams IV or by mouth daily until the patient resumes a normal diet.

Magnesium sulfate

Chronic alcohol use causes the kidneys to waste magnesium. Poor diet means little magnesium comes in. Low magnesium—low magnesium (hypomagnesemia), serum level below 1.7 milligrams per deciliter—makes withdrawal worse. Magnesium normally blocks NMDA glutamate receptors, acting like a natural brake on excitation. It is required for hundreds of enzymes, including those that make ATP and regulate potassium and calcium channels. Without magnesium, brain cells are more excitable. The heart rhythm becomes unstable—risk of torsades de pointes, a deadly arrhythmia. Muscles cramp and twitch. Potassium cannot stay inside cells, so potassium levels drop no matter how much you replace. Magnesium sulfate 2 grams IV over 15 minutes, then 1 to 2 grams every 6 hours until serum level is above 2 milligrams per deciliter, replaces the shortage. Oral magnesium oxide, 400 milligrams once or twice daily, continues replacement after the acute phase.

Potassium chloride

Alcohol causes the kidneys to lose potassium. Vomiting or diarrhea makes it worse. Low potassium—low potassium (hypokalemia), level below 3.5 milliequivalents per liter—destabilizes the heart's electrical system. This causes arrhythmias, especially dangerous when the autonomic storm of withdrawal is already stressing the heart. Potassium is the main positive ion inside cells. The gradient—difference in concentration—between inside and outside the cell determines how easily brain cells and heart muscle cells can fire and recover. Low potassium also weakens skeletal muscles and slows gut movement. Replace with potassium chloride 10 to 20 milliequivalents per hour IV—never faster, or it can stop the heart. The target is a serum level above 4 milliequivalents per liter. In less urgent cases, give oral potassium chloride 20 to 40 milliequivalents two to four times daily.

Sodium chloride and dextrose (IV fluids)

Patients in alcohol withdrawal are often dehydrated from poor intake, vomiting, sweating, and fever. Dehydration drops blood volume. This stresses the heart and kidneys and makes low blood pressure worse when sedatives are given. Normal saline—0.9% sodium chloride—or lactated Ringer's solution replaces lost fluid and sodium. After thiamine is given, add dextrose—5% dextrose in half-normal saline—to provide glucose, the brain's only fuel. Chronic drinkers often have low blood sugar—low blood sugar (hypoglycemia)—from poor nutrition and depleted liver glycogen stores. Typical starting rate: 100 to 150 milliliters per hour IV. Adjust based on urine output, blood pressure, and heart rate. The goal is urine output above 0.5 milliliters per kilogram per hour and stable blood pressure without needing vasopressors.

Haloperidol

Haloperidol blocks dopamine D2 receptors in the brain. This can calm severe agitation and hallucinations. In alcohol withdrawal, the main problem is too much glutamate and too little GABA. But some patients also have very high dopamine activity. Dopamine is a brain messenger involved in reward, movement, and mood. Very high dopamine worsens paranoia, visual hallucinations, and violent agitation. Haloperidol is used ONLY as an add-on to benzodiazepines, never alone. It does NOT fix the GABA-glutamate imbalance. It can actually LOWER the seizure threshold—make seizures more likely. Typical dose: 0.5 to 5 milligrams IV or IM—into the muscle—every 30 to 60 minutes as needed for agitation that persists despite adequate sedation with benzodiazepines. Use the lowest effective dose for the shortest time.

Folic acid (vitamin B9)

Chronic alcohol use drains folic acid because alcohol blocks its absorption in the small intestine. Heavy drinkers often eat poorly. Folic acid is needed to make new DNA and RNA when cells divide. It is required to convert homocysteine—a potentially toxic amino acid—into methionine—an amino acid and methyl donor. Without folic acid, red blood cells grow too large and do not divide properly. This causes macrocytic anemia—low red blood cell count with large, fragile cells. Brain cells also need folic acid to maintain myelin—the insulation around nerves—and to produce neurotransmitters. Replacing folic acid with 1 milligram by mouth or IV daily prevents worsening anemia and supports brain recovery. Folic acid is often given alongside thiamine because both are commonly depleted together.

Phosphate (sodium phosphate or potassium phosphate)

Chronic alcohol use and poor nutrition often cause low phosphate—hypophosphatemia, level below 2.5 milligrams per deciliter. Phosphate is essential for making ATP—the energy currency of cells. It is part of the backbone of DNA and RNA. Severe low phosphate—below 1.0 milligram per deciliter—causes muscle weakness, including the diaphragm. This risks breathing failure. It causes rhabdomyolysis—muscle breakdown releasing myoglobin that clogs

the kidneys. It causes red blood cell destruction (hemolysis)—red blood cells rupture—and heart failure. In alcohol withdrawal, the stress and high energy demand make low phosphate especially dangerous. Replace with sodium phosphate or potassium phosphate 0.16 to 0.32 millimoles per kilogram IV over 6 hours. Target a serum level above 2.5 milligrams per deciliter. Oral replacement—Neutra-Phos or K-Phos 1 to 2 packets or tablets three times daily—is safer for mild shortage.

Where this information comes from

- ASAM Clinical Practice Guideline on Alcohol Withdrawal Management, 2020
- APA Practice Guideline for the Treatment of Patients with Substance Use Disorders, 2nd Edition
- Cochrane Database Systematic Review: Benzodiazepines for Alcohol Withdrawal, 2019
- NEJM Clinical Practice: Alcohol Withdrawal, 2014

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